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JAUNDICE ASSOCIATED WITH HYPERTROPHIC PYLORIC STENOSIS

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The association of jaundice with hypertrophic pyloric stenosis appears to be rare but may result in confusion in management of the infant because it directs attention to possible lesions in the liver or extrahepatic biliary tract. That the association is a valid one, but not a clinically serious one, is evidenced by the rapid disappearance of jaundice following pyloromyotomy. The jaundice is characterized by an indirect hyperbilirubinemia.

In addition to its importance in interpreting the clinical condition of the patient, the association has been of interest because of the difficulty in explaining the etiology of the jaundice. Characteristically, the "physiologic jaundice" of the newborn period clears, to be followed by the second-phase hyperbilirubinemia associated with the features of pyloric obstruction due to the hypertrophic pyloric stenosis. The experience of others\(^1,2\) and the patient herein reported indicate that the hyperbilirubinemia is not due to any microscopically identifiable liver abnormality nor to mechanical obstruction to the flow of the bile within the liver or in the extrahepatic biliary tract. The level of bilirubin has not reached levels which would produce cerebral damage and ordinarily occurs beyond the age when the young brain is susceptible to kernicterus.

ABSTRACT OF PATIENT'S COURSE

A three and one-half week old infant was hospitalized because of jaundice, and vomiting of three days duration. The birth was full term; a breech delivery was required. Jaundice had been noted during the first several days of life and then subsided. The weight pattern for the infant had not been unusual but a weight loss of seven ounces had occurred following the onset of vomiting. A male sibling required operation for hypertrophic stenosis two years previously.

At the time of admission to the hospital the baby was visibly icteric. The upper abdomen was distended; there was visible peristalsis. A mass was palpable in the right upper abdomen. Vomiting occurred immediately after liquid intake. The total bilirubin was 14.4 mgm. per cent of which only 1.86 mgm. per cent was of the direct reacting component. The serum glutamic oxaloacetic transaminase (SGOT) was 60 units. Bile was present in the urine. The urine urobilinogen and a direct Coombs' test were negative. The blood hemoglobin, leucocyte count, serum electrolyte studies, and the prothrombin time were within normal limits.

On the day following admission, a pyloromyotomy was done using local anesthesia. The liver, gallbladder, and extrahepatic biliary tract were normal to gross examination. A cholangiogram obtained by injecting radioopaque media into the gallbladder showed no

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abnormalities in the extrahepatic ducts with good flow into the duodenum (Figure 1). A liver biopsy was taken which was considered to show no histologic abnormalities. Much glycogen was noted in the liver cells and a moderate amount of iron was also present. No evidence of bile stasis was seen.

The infant had a satisfactory postoperative course and the jaundice rapidly disappeared. On the sixth postoperative day the total serum bilirubin was 1.69 mgm. per cent of which 0.61 mgm. per cent was of the direct reacting variety. The child has had no subsequent difficulty except for an episode of pneumonia at the age of 14 months. Another male sibling born subsequently had no difficulty.

**DISCUSSION**

A number of case reports have appeared in the medical literature, especially in the French journals, since Martin and Siebenthal in 1955 apparently first called attention to the association of jaundice with hypertrophic pyloric stenosis. The etiology of the jaundice and the relationship between the two conditions remains conjectural. The reason that attention was not called to this association prior to 1955 is also uncertain.

Although mechanical factors of compression of extrahepatic bile ducts or vessels have been introduced as possible pathogenetic reasons for the jaundice, the presence of jaundice is more adequately explained by a deficiency in conjugation of bilirubin in the hepatic cells. A definite decrease in glucuronyl transferase activity in the liver cells of one infant has been reported.

If the jaundice associated with pyloric stenosis is due to a defect in conjugation of bilirubin in the liver, the reason for the deficiency and its relation to pyloric stenosis should be explained. The disturbance in gastric function (delayed gastric emptying, gastric hyperperistalsis) may aggravate immaturity of hepatic cell function, may actually inhibit cellular function of the liver, or may interfere with assimilation in the digestive tract of a substance such as glucose which is essential for conjugation of bilirubin. A combination of factors may be involved.
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The severity of icterus and variety of bilirubin present in infants presenting jaundice associated with pyloric stenosis are similar to the “physiologic” jaundice of newborn. The enzyme transferring the glucuronyl radical to bilirubin has been found to be absent or deficient in human neonatal jaundice. A genetic enzymatic defect would not fully explain the disturbance, since it does not account for the close relationship of jaundice to the presence of pyloric stenosis.

Irrespective of the etiology of the jaundice associated with pyloric stenosis, the occurrence of this relationship and the rapid relief produced by pyloromyotomy should be recognized. Proper medical and surgical therapy for pyloric stenosis continues to produce excellent results.

SUMMARY AND CONCLUSIONS

1. A patient demonstrating the association of jaundice with pyloric stenosis is reported.

2. It is important to recognize that this condition is not due to extrahepatic biliary tract obstruction and clears rapidly after pyloromyotomy. The elevated bilirubin is predominantly of the indirect variety.

3. The etiology of the jaundice in these patients has not been explained. It appears most likely to be the result of a deficiency in the glucuronide conjugating system in the liver cell.

REFERENCES


